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APPLICATION NO.	FILING DATE	FIRST NAMED INVENTOR	ATTORNEY DOCKET NO.	CONFIRMATION NO.
09/975,856	10/11/2001	Ali O. Gurc	LUD-5480.2 DIV	2513
24972	7590	05/11/2004	EXAMINER	
FULBRIGHT & JAWORSKI, LLP 666 FIFTH AVE NEW YORK, NY 10103-3198			UNGAR, SUSAN NMN	
			ART UNIT	PAPER NUMBER

1642

DATE MAILED: 05/11/2004

Please find below and/or attached an Office communication concerning this application or proceeding.

Office Action Summary	Application No. 09/975,856	Applicant(s) GURE ET AL.	
	Examiner Susan Ungar	Art Unit 1642	

-- The MAILING DATE of this communication appears on the cover sheet with the correspondence address --

Period for Reply

A SHORTENED STATUTORY PERIOD FOR REPLY IS SET TO EXPIRE 3 MONTH(S) FROM THE MAILING DATE OF THIS COMMUNICATION.

- Extensions of time may be available under the provisions of 37 CFR 1.136(a). In no event, however, may a reply be timely filed after SIX (6) MONTHS from the mailing date of this communication.
- If the period for reply specified above is less than thirty (30) days, a reply within the statutory minimum of thirty (30) days will be considered timely.
- If NO period for reply is specified above, the maximum statutory period will apply and will expire SIX (6) MONTHS from the mailing date of this communication.
- Failure to reply within the set or extended period for reply will, by statute, cause the application to become ABANDONED (35 U.S.C. § 133). Any reply received by the Office later than three months after the mailing date of this communication, even if timely filed, may reduce any earned patent term adjustment. See 37 CFR 1.704(b).

Status

- 1) ☐ Responsive to communication(s) filed on 27 February 2004 and 01 March 2004.
- 2a) ☐ This action is **FINAL**. 2b) ☒ This action is non-final.
- 3) ☐ Since this application is in condition for allowance except for formal matters, prosecution as to the merits is closed in accordance with the practice under *Ex parte Quayle*, 1935 C.D. 11, 453 O.G. 213.

Disposition of Claims

- 4) ☐ Claim(s) 25-30 is/are pending in the application.
- 4a) Of the above claim(s) _____ is/are withdrawn from consideration.
- 5) ☐ Claim(s) _____ is/are allowed.
- 6) ☐ Claim(s) 25-30 is/are rejected.
- 7) ☐ Claim(s) _____ is/are objected to.
- 8) ☐ Claim(s) _____ are subject to restriction and/or election requirement.

Application Papers

- 9) ☐ The specification is objected to by the Examiner.
- 10) ☐ The drawing(s) filed on _____ is/are: a) ☐ accepted or b) ☐ objected to by the Examiner.
Applicant may not request that any objection to the drawing(s) be held in abeyance. See 37 CFR 1.85(a).
Replacement drawing sheet(s) including the correction is required if the drawing(s) is objected to. See 37 CFR 1.121(d).
- 11) ☐ The oath or declaration is objected to by the Examiner. Note the attached Office Action or form PTO-152.

Priority under 35 U.S.C. § 119

- 12) ☐ Acknowledgment is made of a claim for foreign priority under 35 U.S.C. § 119(a)-(d) or (f).
- a) ☐ All b) ☐ Some * c) ☐ None of:
1. ☐ Certified copies of the priority documents have been received.
 2. ☐ Certified copies of the priority documents have been received in Application No. _____.
 3. ☐ Copies of the certified copies of the priority documents have been received in this National Stage application from the International Bureau (PCT Rule 17.2(a)).

* See the attached detailed Office action for a list of the certified copies not received.

Attachment(s)

- | | |
|--|---|
| 1) <input checked="" type="checkbox"/> Notice of References Cited (PTO-892) | 4) <input type="checkbox"/> Interview Summary (PTO-413)
Paper No(s)/Mail Date. _____ |
| 2) <input type="checkbox"/> Notice of Draftsperson's Patent Drawing Review (PTO-948) | 5) <input type="checkbox"/> Notice of Informal Patent Application (PTO-152) |
| 3) <input type="checkbox"/> Information Disclosure Statement(s) (PTO-1449 or PTO/SB/08)
Paper No(s)/Mail Date _____ | 6) <input type="checkbox"/> Other: _____ |

1. The Election filed February 27 in response to the Office Action of February 17, 2004 and the Supplementary Election March 1, 2004 are acknowledged and have been entered. Claims 1-24 were cancelled and new claims 25-30 have been added. Claims 25-30 are currently under prosecution.

Specification

2. The specification on page 1 should be amended to reflect the status of the parent applications.

Claim Rejections - 35 USC 101

3. 35 U.S.C. 101 reads as follows:

"Whoever invents or discovers any new and useful process, machine, manufacture, or composition of matter or any new and useful improvement thereof, may obtain a patent therefor, subject to the conditions and requirements of this title".

4. Claims 25-30 are rejected under 35 U.S.C. 101 because the claimed invention is not supported by either a substantial utility or a well established utility.

The disclosed utilities for a method for determining expression of an SSX4 gene/cancer/melanoma and a method of determining expression of an SSX5 gene/cancer/melanoma comprising determining expression of an SSX4 gene/SSX5 gene comprising contacting a sample with an isolated antibody which specifically binds to a protein encoded by SSX4 gene/SSX5 gene appears to be their use as diagnostic markers for detection of cancer/melanoma (p. 2). The specification teaches that the relationship between some tumor associated genes and the SSX genes is under investigation wherein SSX2 was identified as a tumor antigen which is present in 70% of synovial sarcomas (p. 5), wherein SSX2 is differentially expressed in a number of tumor cells and in normal testes only compared to normal control (p. 6) wherein homologs of SSX2, that is SSX4 and SSX5, were identified,

cloned (p. 6) and isolated (p. 8), wherein SSX1 and SSX3 were also cloned and isolated (p. 6) (it is noted that whereas SSX1 mRNA was shown by Crew et al (EMBO J., 1995, 14(2333-2340) to be useful for the diagnosis of synovial sarcoma (see abstract), SSX3 mRNA was shown to be not implicated in synovial sarcomas (see abstract, de Leeuw et al Cytogenetic and Genome Research, 1996, 73:179-183) and thus not useful for diagnosing synovial sarcomas), wherein the SSX4 polynucleotide exists in two forms, the wild-type represented by SEQ ID NO:1 and a splice variant of SSX4 (p. 11) which, due to alternate splicing and shifting of a downstream open reading frame shows no homology to the other SSX proteins. It is noted that SSX4 has 79.8% homology to SSX2, a known tumor antigen and that SSX5 has a 83.5% homology to SSX2 a known tumor antigen.

However, neither the specification nor any art of record teaches what SSX4 or SSX5 is, what they do, they do not teach a utility for the claimed splice variant of SSX4, do not teach a relationship to any specific diseases or establish any involvement in the etiology of any specific diseases for the claimed species. The asserted utility of SSX4 and SSX5 appears to be based on the assertion that the SSX4 and SSX5 have sequence identity to SSX2, a known tumor antigen. However, there is no showing that either SSX4 or SSX5 mRNA or protein is differentially expressed in any primary tumors as compared with normal controls. Thus, although SSX2 appears to be a tumor antigen, the activity of this antigen cannot be extrapolated to other SSX genes or their protein products and the claimed invention does not have a well established utility because it is unknown, in the absence of further experimentation, whether or not SSX4 or SSX5 is a tumor antigen. Given this, the claimed invention does not have substantial utility since

additional experimentation is required in order to establish a real world use for the claimed invention.

In addition, although the specification clearly discloses that SSX4 and SSX5 mRNA are expressed in a single cell line that expresses SSX2, a review of Table 3 reveals that neither SSX4 nor SSX5 mRNA is expressed in any of the other 11 cell lines assayed although SSX2 is expressed in two of those cell lines. It is noted that the cell lines were not assayed for the SSX4 splice variant that encodes a polypeptide without homology to other SSX encoded proteins. It is clear that these cell lines cannot be used to determine whether the expression of a particular gene mRNA in the cell culture environment is in any way related to the *in vivo* cells from which the cell lines were derived in view of the art recognized problems with artifacts associated with cell culture. For example, Drexler et al (Leukemia and Lymphoma, 1993, 9:1-25) specifically teach, in the study of Hodgkin and Reed-Sternberg cancer cells in culture, that the acquisition or loss of certain properties during adaptation to culture systems cannot be excluded. This is exemplified by the teachings of Zellner et al (Clin. Can. Res., 1998, 4:1797-17802) who specifically teach that products are overexpressed in glioblastoma (GBM)-derived cell lines which are not overexpressed *in vivo*. Drexler et al further teach that only a few cell lines containing cells that resemble the *in-vivo* cancer cells have been established and even for the bona fide cancer cell lines it is difficult to prove that the immortalized cells originated from a specific cancer cell (see attached abstract). Further, Embleton et al (Immunol Ser, 1984, 23:181-207) specifically teaches that in procedures for the diagnosis of osteogenic sarcoma, caution must be used when interpreting results obtained with monoclonal antibodies that had been raised to cultured cell lines and specifically teach that

cultured tumor cells may not be antigenically typical of the tumor cell population from which they were derived and it is well established that new artifactual antigens can occur as a result of culture (see attached abstract). Hsu (in Tissue Culture Methods and Applications, Kruse and Patterson, Eds, 1973, Academic Press, NY, see abstract, p.764) specifically teaches that it is well known that cell cultures *in vitro* frequently change their chromosomal constitutions (see abstract). Given the above, it is clear that additional research is required in order to determine whether SSX4, splice variant or SSX5 mRNA is differentially expressed in primary tumor cells compared to normal controls and the invention does not have substantial utility.

Further, the claims are drawn to a method of determining expression of an SSX4 gene/SSX5 gene by determining the binding of an antibody to a polypeptide encoded by said gene or the SSX4 splice variant. However, there is no showing that a polypeptide, encoded by either of these polynucleotides, is produced *in vivo*. In particular, it is well known in the art that the regulation of mRNA translation is one of the major regulatory steps in the control of gene expression (Jansen, et al, 1995, Pediatric Res., 37(6):681-686). Further, those of skill in the art, recognize that expression of mRNA, specific for a tissue type, does not dictate nor predict the translation of such mRNA into a polypeptide. For example, Alberts et al. (Molecular Biology of the Cell, 3rd edition, 1994, page 465) teach that translation of ferritin mRNA into ferritin polypeptide is blocked during periods of iron starvation. Likewise, if excess iron is available, the transferrin receptor mRNA is degraded and no transferrin receptor polypeptide is translated. Many other proteins are regulated at the translational level rather than the transcriptional level. For instance, Shantz and Pegg (Int J of Biochem and Cell Biol., 1999, Vol. 31, pp. 107-

122) teach that ornithine decarboxylase is highly regulated in the cell at the level of translation and that translation of ornithine decarboxylase mRNA is dependent on the secondary structure of the mRNA and the availability of eIF-4E, which mediates translation initiation. McClean and Hill (Eur J of Cancer, 1993, vol. 29A, pp. 2243-2248) teach that p-glycoprotein can be overexpressed in CHO cells following exposure to radiation, without any concomitant overexpression of the p-glycoprotein mRNA. In addition, Fu et al (EMBO Journal, 1996, Vol. 15, pp. 4392-4401) teach that levels of p53 protein expression do not correlate with levels of p53 mRNA levels in blast cells taken from patients with acute myelogenous leukemia, said patients being without mutations in the p53 gene. Thus, protein translation is not necessarily contingent on mRNA expression due to the multitude of homeostatic factors affecting transcription and translation. Therefore, it cannot be determined, in the absence of further experimentation, if the encoded polypeptides are in actuality expressed in vivo or that they could be used in a binding assay for determining the expression of an SSX4/SSX5 gene and additional experimentation is required in order to determine a real world use for the claimed method and thus the method does not have substantial utility.

Finally, as drawn to the claims which read on the use of the claimed method for the diagnosis of cancer/melanoma, evidence abounds in which protein levels do not correlate with steady-state mRNA levels or alterations in mRNA levels. For instance, Brennan et al (Journal of Autoimmunity, 1989, vol. 2 suppl., pp. 177-186) teach that high levels of the mRNA for TNF alpha were produced in synovial cells, but that levels of the TNF alpha protein were undetectable. Zimmer (Cell Motility and the Cytoskeleton, 1991, vol. 20, pp. 325-337) teaches that there is no correlation between the mRNA level of calcium-modulated protein S100 alpha and

the protein level, indicating that S100 protein is post-transcriptionally regulated. Eriksson et al (*Diabetologia*, 1992, vol. 35, pp. 143-147) teach that no correlation was observed between the level of mRNA transcript from the insulin-responsive glucose transporter gene and the protein encoded thereby. Hell et al (*Laboratory Investigation*, 1995, Vol. 73, pp. 492-496) teach that cells in all types of Hodgkin's disease exhibited high levels of bcl-2 mRNA, while the expression of the Bcl-2 protein was not homogenous to said cells. Powell et al (*Pharmacogenesis*, 1998, Vol. 8, pp. 411-421) teach that mRNA levels for cytochrome P450 E1 did not correlate with the level of corresponding protein, and conclude that the regulation of said protein is highly complex. Carrere et al (*Gut*, 1999, vol. 44, pp. 55-551) teach an absence of correlation between protein and mRNA levels for the Reg protein. Vallejo et al (*Biochimie*, 2000, vol. 82, pp. 1129-1133) teach that no correlation was found between NRF-2 mRNA and protein levels suggesting post-transcriptional regulation of NRF-2 protein levels. Guo et al (*Journal of Pharmacology and Experimental Therapeutics*, 2002, vol. 300, pp. 206-212) teach that Oatp2 mRNA levels did not show a correlation with Oatp2 protein levels, suggesting that regulation of the Oatp2 protein occurs at both the transcriptional and post-translational level. These references serve to demonstrate that levels of polypeptidess cannot be relied upon to anticipate levels of mRNA. Further, Jang et al (*Clinical and Experimental Metastasis*, 1997, vol. 15, pp. 469-483) teach that further studies are necessary to determine if changes in protein levels track with changes in mRNA levels for metastasis associated genes in murine tumor cells, thus providing further evidence that one of skill in the art cannot anticipate that the level of a specific mRNA expressed by a cell will be paralleled at the protein level due to complex homeostatic factors controlling translation and post-translational

modification. Thus additional research must be done in order to establish a real world use for the claimed invention and the invention does not have substantial utility. In summary, the specification relies upon homology to a known tumor antigen to infer that the claimed invention would be useful for diagnosing cancer/melanoma, given the above, the claimed invention has neither a well-established nor a substantial utility, the specification relies on cell culture studies that demonstrate the unpredictability of the expression of SSX4 and SSX5 mRNA in cultured melanoma cells, wherein such findings as the specification presents cannot be correlated to the *in vivo* condition or to the ability to diagnose any cancer for the reasons set forth above, thus the invention does not have substantial utility. In addition, the specification claims that the expression of the gene/cancer/melanoma diagnosis can be determined by contacting the protein expression product of that gene with an antibody wherein it is unknown if any protein is expressed *in vivo* by the claimed genes and the unpredictability of the correlation between mRNA synthesis and protein expression is well known in the art. The specification essentially gives an invitation to experiment wherein the artisan is invited to elaborate a functional use for the claimed methods. Because the claimed invention is not supported by either a well-established or a substantial utility for the reasons set forth, credibility of any utility cannot be assessed.

5. Claims 28-30 are rejected under 35 U.S.C. 101 because the disclosed invention is inoperative and therefore lacks utility. The invention is inoperative because it is drawn to a method for determining expression of an SSX4 gene by contacting a sample with an isolated antibody which specifically binds to a protein encoded by an isolated nucleic acid molecule having the nucleotide sequence of SEQ ID NO:2 which encodes the SSX5 protein gene product.

Claim Rejections - 35 USC 112

6. 35 U.S.C. 112 reads as follows:

"The specification shall contain a written description of the invention, and of the manner and process of making and using it, in such full, clear, concise, and exact terms as to enable any person skilled in the art to which it pertains, or with which it is most nearly connected, to make and use the same and shall set forth the best mode contemplated by the inventor of carrying out his invention."

7. Claims 25-30 are rejected under 35 U.S.C. 112, first paragraph.

Specifically, since the claimed invention is not supported by a substantial utility, a well established utility for the reasons set forth in the rejection under 35 USC 101 above, one skilled in the art clearly would not know how to use the claimed invention.

8. In the event that Applicants might be able to overcome the 35 USC 101 rejection above, Claims 25-30 would still be rejected under 35 USC 112, first paragraph because the specification, while being enabling for the claimed method comprising contacting a sample with an isolated antibody which specifically binds to a protein encoded by a nucleic acid sequence which consists of SEQ ID NO:1, the claimed splice variant thereof or SEQ ID NO:2 does not reasonably provide enablement for said method comprising contacting a sample with an isolated antibody which specifically binds to a protein encoded by a nucleic acid sequence having the nucleotide sequence of SEQ ID NO:1, the claimed splice variant of SEQ ID NO:1 or encoded by a nucleic acid sequence having the nucleotide sequence of SEQ ID NO:2. The specification does not enable any person skilled in the art to which it pertains, or with which it is most nearly connected, to use the invention commensurate in scope with these claims.

The claims are drawn to the claimed method wherein the determination of expression is done by assaying with an antibody which specifically binds to a protein encoded by a nucleic acid sequence having the nucleotide sequence of SEQ ID NO:1, the claimed splice variant of SEQ ID NO:1 or encoded by a nucleic acid sequence having the nucleotide sequence of SEQ ID NO:2. Given the open meaning of the term “having” which is read by the Office as equivalent to the term “comprising”, the claims are clearly drawn to antibodies that do not bind to and are not specific for the proteins encoded by SEQ ID NO:1, the splice variant thereof or to SEQ ID NO:2. The specification teaches as set forth above. One cannot extrapolate the teaching of the specification to the scope of the invention because the scope of the invention includes antibodies that do not bind to and are not specific for the proteins encoded by SEQ ID NO:1, the splice variant thereof or to SEQ ID NO:2 and in the absence of that specificity one would not know how to use the claimed invention. The specification provides insufficient guidance with regard to this issue and provides no working examples which would provide guidance to one skilled in the art and no evidence has been provided which would allow one of skill in the art to predict that invention would function as claimed with a reasonable expectation of success. For the above reasons, it appears that undue experimentation would be required to practice the claimed invention. The rejection can be obviated by amending claims 25 and 28, for example, to delete the term “having” and substitute the phrase “consisting of”.

9. In the event that Applicants might be able to overcome the 35 USC 101 and 35 USC 112, first paragraph rejections above, Claims 26, 29 would still be rejected under 35 USC 112, first paragraph because the specification, while being enabling for a method for determining expression of an SSX4/SSX5 gene by assaying

encoded protein wherein the encoded protein indicates the presence of melanoma does not reasonably provide enablement for a method for determining expression of an SSX4/SSX5 gene by assaying encoded protein wherein the encoded protein indicates the presence of cancer. The specification does not enable any person skilled in the art to which it pertains, or with which it is most nearly connected, to use the invention commensurate in scope with these claims.

The claims are drawn to a method for determining expression of an SSX4/SSX5 gene comprising contacting a sample with an isolated antibody which specifically binds to a polypeptide encoded by a molecule having the nucleotide sequence of SEQ ID NO:1, a splice variant of SEQ ID NO:1 or SEQ ID NO:2. This means that the antibody is not required to bind to a protein encoded by a molecule consisting of the nucleotide sequence of SEQ ID NO:1, a splice variant of SEQ ID NO:1 or SEQ ID NO:2. The specification teaches that the relationship between some tumor associated genes and the SSX genes is under investigation wherein SSX2 was identified as a tumor antigen which is present in 70% of synovial sarcomas (p. 5), wherein SSX2 is differentially expressed in a number of tumor cells and in normal testes only compared to normal control (p. 6) wherein homologs of SSX2, that is SSX4 and SSX5, were identified, cloned (p. 6) and isolated (p. 8), wherein SSX1 and SSX3 were also cloned and isolated (p. 6) (it is noted that whereas SSX1 mRNA was shown by Crew et al (EMBO J., 1995, 14(2333-2340) to be useful for the diagnosis of synovial sarcoma (see abstract), SSX3 mRNA was shown by to be not implicated in synovial sarcomas (see abstract, de Leeuw et al Cytogenetic and Genome Research, 1996, 73:179-183) and thus not useful for diagnosing synovial sarcomas), wherein the SSX4 polynucleotide exists in two forms, the wild-type represented by SEQ ID NO:1 and

a splice variant of SSX4 (p. 11) which, due to alternate splicing and shifting of a downstream open reading frame shows no homology to the other SSX proteins. It is noted that SSX4 has 79.8% homology to SSX2, a known tumor antigen and that SSX5 has a 83.5% homology to SSX2 a known tumor antigen. One cannot extrapolate the teaching of the specification to the scope of the claims because it is clear from the information in the specification that not all SSX genes are expressed in all tumors. Although it is clear that SSX2 and SSX1 mRNA are expressed and are diagnostic of synovial sarcoma (see above), it is equally clear that SSX3 is not. Given the art recognized heterogeneity of tumors, given the information in the application as originally filed, it cannot be predicted and no one of skill in the art would believe that it is more likely than not that SSX4 or SSX5 gene is differentially expressed in any cancer other than melanoma based only on the homology of the two genes to SSX2, a known tumor antigen, or that given the information set forth above, the polypeptide encoded by the claimed sequences would be differentially expressed and would serve as a diagnostic marker for any cancer other than melanoma. The specification provides insufficient guidance with regard to these issues and provides no working examples which would provide guidance to one skilled in the art and no evidence has been provided which would allow one of skill in the art to predict that invention would function as claimed with a reasonable expectation of success. For the above reasons, it appears that undue experimentation would be required to practice the claimed invention.

Double Patenting

10. The non-statutory double patenting rejection, whether of the obviousness type or non-obviousness type, is based on a judicially created doctrine grounded in public policy (a policy reflected in the statute) so as to prevent the unjustified or

improper timewise extension of the "right to exclude" granted by a patent. *In re Thorington*, 418 F.2d 528, 163 USPQ 644 (CCPA 1969); *In re Vogel*, 422 F.2d 438, 164 USPQ 619 (CCPA 1970); *In re Van Ornam*, 686 F.2d 937, 214 USPQ 438, 761 (CCPA 1982); *In re Longi*, 759 F.2d 887, 225 USPQ 645 (Fed. Cir. 1985); and *In re Goodman*, 29 USPQ2d 2010 (Fed. Cir. 1993).

A timely filed terminal disclaimer in compliance with 37 CFR 1.321 (b) and (c) may be used to overcome an actual or provisional rejection based on a non-statutory double patenting ground provided the conflicting application or patent is shown to be commonly owned with this application. See 37 CFR 1.78 (d).

Effective January 1, 1994, a registered attorney or agent of record may sign a terminal disclaimer. A terminal disclaimer signed by the assignee must fully comply with 37 CFR 3.73(b)

Claims 25, 26 and 28-29 are rejected under the judicially created doctrine of obviousness-type double patenting as unpatentable over 1, 5, 7, 20 of US Patent No. 6,287,756. Although the conflicting claims are not identical, they are not patentably distinct from each other because they relate to the same inventive concept, that is to a method for determining expression of an SSX4 gene/SSX5 gene wherein expression of said gene is indicative of cancer in a subject wherein determination is made by determining protein encoded by a nucleic acid molecule having the nucleotide sequence of SEQID NO:1, SEQ ID NO:2. Although the issued patent does not specifically claim assaying with an antibody specific to said encoded protein, assaying the protein expressed by the claimed SSX gene with an antibody would have been *prima facie* obvious at the time the invention was made since antibody detection of proteins was conventional in the art.

11. No claims allowed.

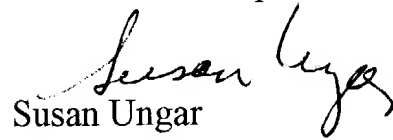
Art Unit: 1642

12. Any inquiry concerning this communication or earlier communications from the examiner should be directed to Susan Ungar, PhD whose telephone number is (571) 272-0837. The examiner can normally be reached on Monday through Friday from 7:30am to 4pm.

If attempts to reach the examiner by telephone are unsuccessful, the examiner's supervisor, Yvonne Eyler, can be reached at (571) 272-0871. The fax phone number for this Art Unit is (703) 872-9306.

Any inquiry of a general nature or relating to the status of this application or proceeding should be directed to the Group receptionist whose telephone number is (703) 308-0196.

Effective, February 7, 1998, the Group and/or Art Unit location of your application in the PTO has changed. To aid in correlating any papers for this application, all further correspondence regarding this application should be directed to Group Art Unit 1642.


Susan Ungar
Primary Patent Examiner
April 30, 2004